CLAIMS

1. A method of treating, preventing or ameliorating chronic heart failure or acute heart failure in a patient the method comprising administering to the patient an effective amount of a compound that is able to reduce the production, absorption and/or the effect of an endotoxin (lipopolysaccharide; LPS).

2. A method of treating, preventing or ameliorating endotoxin-mediated immune activation in acute or chronic heart failure in a patient the method comprising administering to the patient an effective amount of a compound that is able to reduce the production, absorption and/or the effect of an endotoxin (lipopolysaccharide; LPS).

3. A method according to claim 1 and 2 wherein the compound is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule.

4. A method according to claim 1 to 3 wherein the compound is able to reduce the available endotoxin in the patient.

5. A method according to claim 1 to 4 wherein the compound is a bile acid.

6. A method according to claim 1 to 5 wherein the bile acid is any one of ursodesoxycholic acid, chemodeoxycholic acid, dehydrocholic acid, cholic acid and deoxycholic acid.

7. A method according to claim 1 to 6 wherein the compound is LPS binding protein, bactericidal/permeability increasing protein (BPI), a lipoprotein, for instance but not exclusively low density lipoprotein (LDL), high density lipoprotein (HDL), very low density lipoprotein (VLDL), apolipoprotein (a), a lipoprotein mixture or an antibody capable of binding to endotoxin (lipopolysaccharide; LPS).

8. A method according to claim 1 and 2 wherein the compound is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule in the gut.

9. A method according to claim 1, 2 and 8 wherein the compound is able to reduce the absorption of endotoxin by the patient from the gut.

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10. A method according to claim 1, 2 and 8, 9 wherein the compound is able to substantially reduce the availability of endotoxin (lipopolysaccharide) for absorption from the gut, such that the amount of endotoxin that is absorbed is reduced or is less biologically active.

11. A method according to claim 1,2 and 8 to 10 wherein the compound is activated charcoal, activated carbon, Fuller's earth, attapulgite, kaolin, bentonite or a clay or colostrum of human, bovine, or other mamallian origin.

12. A method according to claim 1 and 2 wherein the compound is an antibacterial agent.

13. A method according to claim 1, 2 and 12 wherein the antibacterial agent is active in the gut.

14. A method according of claim 1, 2 and 12, 13 wherein the antibacterial agent is able to substantially reduce the amount of bacteria and/or free endotoxin (lipopolysaccharide) in the gut.

15. A method according of claim 1, 2 and 12 to 14 wherein the antibacterial agent is largely unabsorbed from the gut.

16. A method according of claim 1, 2 and 12 to 15 wherein the antibacterial agent is an antibiotic, for instance but not exclusively non-absorbable antibiotics like neomycin, tobramycin, amphotericin B, and colistin.

17. A method according to claim 1 and 2 wherein the compound is able to inhibit the response by a cell to endotoxin (lipopolysaccharide; LPS).

18. A method according to claim 1, 2 and 17 wherein the compound is able to decrease the cytokine production by a cell in response to endotoxin (lipopolysaccharide; LPS).

19. A method according to claim 1, 2 and 17, 18 wherein the compound is an antibody able to bind the CD14 receptor, soluble CD14 receptor or an antibody or non-functional agonist of a toll-like receptor, particularly toll-like receptor 4 and 2.

20. A method according to claim 1, 2 and 17 to 19 wherein the compound is able to inhibit signalling *via* the CD14 receptor or via a toll-like receptor, particularly toll-like receptor 4 and 2.

21. A method according to claim 1 and 2 wherein the compound is able to reduce the permeability of the gut wall to bacteria and/or endotoxin (lipopolysaccharide; LPS).

22. A method according to claim 1, 2 and 21 wherein the agent is able to reduce the amount of bacteria and/or free endotoxin (lipopolysaccharide) that is able to translocate from the gut into the circulation of the patient.

23. A method according to claim 1, 2 and 21, 22 wherein the agent is largely unabsorbed from the gut.

24. A method according to claim 1, 2 and 21 to 23 wherein the agent is IGF-1, allopurinol, oxipurinol, or any other unspecific xanthine oxidase inhibitor, or a specific xanthine oxidase inhibitor (like TMX-67), liquorice or its derivatives, for example carbenoxolone, an alginate, sulfacrate or an agent that may form a hydrogel.

25. A method according to any one of the preceding claims wherein the compound is administered orally.

26. A method according to any one of the preceding claims wherein the compound is administered intravenously.

27. A method according to any one of the preceding claims wherein the compound is administered rectally.

28. Use of a compound that is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule in the manufacture of a medicament for treating, preventing or ameliorating chronic heart failure or acute heart failure in a patient.

29. Use of a compound that is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule in the manufacture of a medicament for treating, preventing or ameliorating endotoxin-mediated immune activation in acute or chronic heart failure in a patient.

30. The use of claim 28 or claim 29 wherein the compound is a bile acid or LPS binding protein or bactericial/permeability increasing protein (BPI), a lipoprotein, for instance but not exclusively low densitiy lipoprotein (LDL), high density lipoprotein (HDL), very low density lipoprotein (VLDL), apolipoprotein (a), a lipoprotein mixture or an antibody capable of binding to endotoxin (lipopolysaccharide; LPS). or an antibody capable of binding to LPS.

31. Use of a compound that is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule in the gut in the manufacture of a medicament for treating, preventing or ameliorating chronic heart failure or acute heart failure in a patient.

32. Use of a compound that is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule in the gut in the manufacture of a medicament for treating, preventing or ameliorating endotoxin-mediated immune activation in acute or chronic heart failure in a patient.

33. The use of claim 31 or claim 32 wherein the compound is activated charcoal, activated carbon, Fuller's earth, attapulgite, kaolin or bentonite or a clay.

34. Use of an antibacterial agent in the manufacture of a medicament for treating, preventing or ameliorating chronic heart failure or acute heart failure in a patient.

35. Use of an antibacterial agent in the manufacture of a medicament for treating, preventing or ameliorating endotoxin-mediated immune activation in acute or chronic heart failure in a patient.

36. The use of claim 34 or claim 35 wherein the compound is a non-absorbable antibiotic, for instance but not exclusively, like neomycin, tobramycin, amphotericin B, and colistin.

37. Use of a compound that is able to inhibit the response by a cell to endotoxin (lipopolysaccharide; LPS) in the manufacture of a medicament for treating, preventing or ameliorating chronic heart failure or acute heart failure in a patient.

38. Use of a compound that is able to inhibit the response by a cell to endotoxin (lipopolysaccharide; LPS) in the manufacture of a medicament for treating, preventing or ameliorating endotoxin-mediated immune activation in acute or chronic heart failure in a patient.

39. The use of claim 37 or claim 38 wherein the compound is an antibody able to bind the CD14 receptor, soluble CD14 receptor or an antibody or non-functional agonist of a toll-like receptor, particularly toll-like receptor 4 and 2..

40. Use of an agent that is able to reduce the permeability of the gut wall to bacteria and/or endotoxin (LPS) in the manufacture of a medicament for treating, preventing or ameliorating chronic heart failure or acute heart failure in a patient.

41. Use of an agent that is able to reduce the permeability of the gut wall to bacteria and/or endotoxin (LPS) in the manufacture of a medicament for treating, preventing or ameliorating endotoxin-mediated immune activation in acute or chronic heart failure in a patient.

42. The use of claim 40 or claim 41 wherein the agent is IGF-1, allopurinol, oxipurinol, or any other unspecific xanthine oxidase inhibitor, or a specific xanthine oxidase inhibitor (like TMX-67), liquorice or its derivatives, for example carbenoxolone, an alginate, sulfacrate or an agent that may form a hydrogel.

43. The method or use of any of the preceding claims wherein a HMG-coenzyme A-reductase inhibitor that is able to increase lipoprotein levels and is not used to lower LDL / cholesterol levels is administered to the patient.

44. The combined application of any method or use of any of the preceding claims in an individual patient.

45. The method or use of any of the preceding claims wherein a diuretic is administered to the patient.

46. A pharmaceutical formulation comprising bile acid or BPI or LPS binding protein, a lipoprotein, for instance but not exclusively like low densitiy lipoprotein (LDL), high density lipoprotein (HDL), very low density lipoprotein (VLDL), apolipoprotein (a), a lipoprotein mixture, or an antibody capable of binding LPS and a diuretic.

47. A pharmaceutical formulation comprising a compound that is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule in the gut and a diuretic.

48. A pharmaceutical formulation comprising an antibacterial agent and a diuretic.

49. A pharmaceutical formulation comprising a compound that is able to inhibit the response by a cell to endotoxin (lipopolysaccharide; LPS) and a diuretic.

50. A pharmaceutical formulation comprising an agent that is able to reduce the permeability of the gut wall to bacteria and/or endotoxin (LPS) and a diuretic.

51. Any novel method of treating, preventing or ameliorating acute or chronic heart failure as herein disclosed.

52. Any novel pharmaceutical composition as herein disclosed.

53. A method of treating or ameliorating body wasting or cachexia in a patient with liver cirrhosis, chronic obstructive pulmonary disease, chronic renal failure, diabetes, rheumatoid arthritis in a patient the method comprising administering to the patient an effective amount of a compound that is able to reduce the production, absorption and/or the effect of an endotoxin (lipopolysaccharide; LPS).

54. A method of treating, preventing or ameliorating endotoxin-mediated immune activation in body wasting or cachexia in a patient with liver cirrhosis, chronic obstructive pulmonary disease, chronic renal failure, diabetes, rheumatoid arthritis the method comprising administering to the patient an effective amount of a compound that is able to reduce the production, absorption and/or the effect of an endotoxin (lipopolysaccharide; LPS).

55. A method according to claim 53 and 54 wherein the compound is able to bind to an endotoxin (lipopolysaccharide; LPS) molecule.

56. A method according to claim 53 to 55 wherein the compound is able to reduce the available endotoxin in the patient.

57. A method according to claim 53 to 56 wherein the compound is a bile acid.

58. A method according to claim 53 to 56 wherein the bile acid is any one of ursodesoxycholic acid, chemodeoxycholic acid, dehydrocholic acid, cholic acid and deoxycholic acid.

59. A method according to claim 53 to 56 wherein the compound is LPS binding protein.

60. A method according to claim 53 to 56 wherein the compound is bactericidal/permeability increasing protein (BPI).

61. A method according to claim 53 to 56 wherein the compound is, a lipoprotein, for instance, low densitiy lipoprotein (LDL), high density lipoprotein (HDL), very low density lipoprotein (VLDL), apolipoprotein (a), a lipoprotein mixture.

62. A method according to claim 53 to 56 wherein the treatment is a combination of a compound according claim 59 and claim 61.

63. A method according to claim 53 to 56 wherein the compound is or an antibody capable of binding to endotoxin (lipopolysaccharide; LPS).

64. A method according to claim 53 to 56 wherein the compound is or an antibody capable of binding to endotoxin (lipopolysaccharide; LPS).

65. A method according to claim 53 to 56 wherein the compound is an antibody able to bind to the CD14 receptor.

66. A method according to claim 53 to 56 wherein the compound is a soluble CD14 receptor.

67. A method according to claim 53 to 56 wherein the compound is a drug blocking effectively signaling through toll-like receptors, for instance toll-like receptor 4 and toll-like receptor 2.

68. A method according to claim 53 to 56 wherein the compound is colostrum of human, bovine, or other mamallian origin.

69. A method according to claim 53 to 56 wherein the compound is able to inhibit the response by a cell to endotoxin (lipopolysaccharide; LPS).

70. A method according to claim 53 to 56 and 69 wherein the compound is able to decrease the cytokine production by a cell in response to endotoxin (lipopolysaccharide; LPS).

71. A method according to claim 53, 54 and 69, and 70 wherein the compound is a compound named in claim 57 to 68.

72. A method according to any one of the preceding claims wherein the compound is administered orally.

Sub A6 73. A method according to any one of the preceding claims wherein the compound is administered intravenously.

74. A method according to any one of the preceding claims wherein the compound is administered rectally.

75. The combined application of any method or use of any of the preceding claims in an individual patient.

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